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L1      159613 S HITOSHI?/AU OR DEMO?/AU OR JENKINS?/AU OR ENGELHARD?/AU OR MO
L2      1097 S MRE11
L3      3 S L1 AND L2
L4      1 DUP REM L3 (2 DUPLICATES REMOVED)
L5      0 S ((ASSAY OR SCREEN) (P) MODULAT?) AND L2
L6      24509 S ((ASSAY OR SCREEN) (P) MODULAT?)
L7      297 S L6 AND "DNA REPAIR"
L8      0 S L7 AND MRE11
L9      14 S L7 AND HELA
L10     0 S L9 AND "SAK"
L11     435 S "SAK"
L12     0 S L11 AND L2
L13     1 S L11 AND "DNA REPAIR"
L14     101 S SAK (S) (PROTEIN OR POLYPEPTIDE OR PEPTIDE)
L15     71 S L14 NOT PY>=2002
L16     29 DUP REM L15 (42 DUPLICATES REMOVED)
L17     25 S RIGEL
L18     0 S L17 AND L2
L19     9 S L11 AND "CELL PROLIFERATION"
L20     5 DUP REM L19 (4 DUPLICATES REMOVED)
L21     266038 S CELL? (W) PROLIFERATION
L22     27 S L21 AND L2
L23     19 DUP REM L22 (8 DUPLICATES REMOVED)
L24     5 S L23 NOT PY>=2002
L25     1738 S RAD50 OR NBS1
L26     39585 S "THYMIDINE INCORPORATION" OR "BRDU INCORPORATION" OR HOESCHT
L27     125323 S HI299 OR MDA-MB OR MCF7 OR A659 OR HELA OR PC3
L28     2510 S P53 (W) (NULL OR WILD)
L29     772 S L2 AND L25
L30     1 S L2 AND L26
L31     51 S L2 AND L27
L32     0 S L2 AND L28
L33     11 S L29 AND "CELL CULTURE"
L34     11 DUP REM L33 (0 DUPLICATES REMOVED)
L35     2 S L34 NOT PY>=2002
L36     0 S L31 AND "CELL CULTURE"
L37     0 S L31 AND L6
L38     0 S L31 AND L1
L39     0 S L31 AND CULTURE
L40     0 S L31 AND L21
L41     1 S L31 AND L21
L42     9 S L31 AND (ANTIBODY OR ANTISENSE OR PEPTIDE OR CIRCULAR)
L43     6 DUP REM L42 (3 DUPLICATES REMOVED)
L44     950 S "COMPLEMENTATION ASSAY"
L45     3 S L44 AND L2
L46     1 DUP REM L45 (2 DUPLICATES REMOVED)
L47     435 S "DNA REPAIR" (2W) ASSAY
L48     0 S L47 AND L2
L49     0 S L47 AND SAK
L50     388 S L47 NOT PY>=2002
L51     1 S L50 AND L26
L52     9 S L50 AND L27
L53     5 DUP REM L52 (4 DUPLICATES REMOVED)
L54     0 S L28 AND L44
L55     0 S L28 AND L47
L56     125 S L28 AND L27
L57     10 S L56 AND L21
L58     7 DUP REM L57 (3 DUPLICATES REMOVED)
L59     4 S L58 NOT PY>=2002
L60     66 S L2 AND (ANTIBODY OR ANTISENSE OR PEPTIDE OR CIRCULAR)
L61     19 S L60 NOT PY>=2002
L62     15 DUP REM L61 (4 DUPLICATES REMOVED)

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L4 ANSWER 1 OF 1 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2000050557 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10581236
TITLE: **Mre11** is essential for the maintenance of
chromosomal DNA in vertebrate cells.
AUTHOR: Yamaguchi-Iwai Y; Sonoda E; Sasaki M S; **Morrison C**
; Haraguchi T; Hiraoka Y; Yamashita Y M; Yagi T; Takata M;
Price C; Kakazu N; Takeda S
CORPORATE SOURCE: Bayer-Chair Department of Molecular Immunology and
Allergology, Faculty of Medicine.
SOURCE: EMBO journal, (1999 Dec 1) 18 (23) 6619-29.
Journal code: 8208664. ISSN: 0261-4189.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF166094
ENTRY MONTH: 200001
ENTRY DATE: Entered STN: 20000209
Last Updated on STN: 20030218
Entered Medline: 20000131

AB Yeast **Mre11** functions with Rad50 and Xrs2 in a complex that has
pivotal roles in homologous recombination (HR) and non-homologous
end-joining (NHEJ) DNA double-strand break (DSB) repair pathways.
Vertebrate **Mre11** is essential. Conditionally, **MRE11**
null chicken DT40 cells accumulate chromosome breaks and die upon
Mre11 repression, showing frequent centrosome amplification.
Mre11 deficiency also causes increased radiosensitivity and
strongly reduced targeted integration frequencies. **Mre11** is,
therefore, crucial for HR and essential in mitosis through its role in
chromosome maintenance by recombinational repair. Surprisingly perhaps,
given the role of **Mre11** in yeast NHEJ, disruption of NHEJ by
deletion of KU70 greatly exacerbates the effects of **MRE11**
deficiency, revealing a significant **Mre11**-independent component
of metazoan NHEJ.

=>

PROCESSING COMPLETED FOR L19
L20 5 DUP REM L19 (4 DUPLICATES REMOVED)

=> d l20 ibib abs total

L20 ANSWER 1 OF 5 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN

ACCESSION NUMBER: 2005062144 EMBASE
TITLE: **Sak**/Plk4 and mitotic fidelity.
AUTHOR: Swallow C.J.; Ko M.A.; Siddiqui N.U.; Hudson J.W.; Dennis J.W.
CORPORATE SOURCE: J.W. Dennis, Samuel Lunenfeld Research Institute, Mount Sinai Hospital, 600 University Ave. R988, Toronto, Ont. M5G 1X5, Canada. dennis@mshri.on.ca
SOURCE: Oncogene, (10 Jan 2005) Vol. 24, No. 2, pp. 306-312.
Refs: 51
ISSN: 0950-9232 CODEN: ONCNES
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 016 Cancer
022 Human Genetics
029 Clinical Biochemistry
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20050224
Last Updated on STN: 20050224

AB **Sak**/Plk4 differs from other polo-like kinases in having only a single polo box, which assumes a novel dimer fold that localizes to the nucleolus, centrosomes and the cleavage furrow. **Sak** expression increases gradually in S through M phase, and **Sak** is destroyed by APC/C dependent proteolysis. **Sak**-deficient mouse embryos arrest at E7.5 and display an increased incidence of apoptosis and anaphase arrest. **Sak**(+/-) mice are haploinsufficient for tumor suppression, with spontaneous tumors developing primarily in the liver with advanced age. During liver regeneration following partial hepatectomy, **Sak**(+/-) hepatocytes display a delay in reaching the first M phase, multipolar spindles, disorganized tissue morphology and loss of acuity for cyclin B1 expression. Similarly, **Sak**(+/-) MEF cells proliferate slowly, and show a high incidence of centrosome hyper-amplification. We suggest that **Sak** provides feedback to cell cycle regulators, and thereby precision to the switch-like transitions of centrosome duplication and exit-from-mitosis. **Sak** binds to p53, and studies are underway to provide a molecular context for the **Sak**-p53 interaction. Animal models of haploinsufficiency and more comprehensive models of cell cycle regulation should contribute to improvements in cancer risk assessment and novel therapies.

L20 ANSWER 2 OF 5 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN

ACCESSION NUMBER: 2003214649 EMBASE
TITLE: Effect of the polylysine based polymeric polypeptides on the growth and chemotaxis of *Tetrahymena pyriformis*.
AUTHOR: Szabo R.; Mezo G.; Hudecz F.; Kohidai L.
CORPORATE SOURCE: F. Hudecz, Research Group of Peptide Chemistry, Hungarian Academy of Science, P.O. Box 32, H-1518, Budapest 112, Hungary. hudecz@szerves.chem.elte.hu
SOURCE: Journal of Bioactive and Compatible Polymers, (2002) Vol. 17, No. 6, pp. 399-415.
Refs: 23
ISSN: 0883-9115 CODEN: JBCPEV
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 027 Biophysics, Bioengineering and Medical Instrumentation
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 20030612
Last Updated on STN: 20030612

AB Polylysine based branched polypeptides represents a group of biocompatible polymers that could be utilized as macromolecular carriers for drugs, epitopes or reporter molecules. Ten polymers with different character (amino acid composition and charge properties) were prepared: polypeptides with single amino acid in the branches (poly[Lys(X(i))]), X=His, Pro or Glu; and polymers possessing oligo[DL-alanine] side chains only (poly[Lys(DL-Ala(m)) (AK) or with an additional amino acid residue poly[Lys(X(i)-DL-Ala(m)) (XAK), where X=Ser (**SAK**), Thr (TAK), Glu (EAK), acetyl-Glu (Ac-EAK) or succinyl-Glu (Succ-EAK). were investigated. The concentration of these compounds influence the chemotaxis and survival of eukaryotic unicellular model organism, Tetrahymena pyriformis GL. Two types of experiments were performed. First the polymer induced chemoattractant/chemorepellent response of Tetrahymena cells were tested, then chemotactic selection experiments were performed. The chemotactic responses elicited by the polymers were dependent not only on chemical properties (composition, charge and the length of the side chain) of the compounds, but also on their concentration. Based on these results, the polymers were grouped as full-chemoattractant expressing this behavior in the full concentration range investigated (H(i)K), full-chemorepellent (E(i)K and Ac-EAK) and partial chemoattractant/chemorepellent with concentration dependent activity (P(i)K, EAK and Succ-EAK).

L20 ANSWER 3 OF 5 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2001569350 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11489907
TITLE: **Sak** serine-threonine kinase acts as an effector of Tec tyrosine kinase.
AUTHOR: Yamashita Y; Kajigaya S; Yoshida K; Ueno S; Ota J; Ohmine K; Ueda M; Miyazato A; Ohya K; Kitamura T; Ozawa K; Mano H
CORPORATE SOURCE: Divisions of Functional Genomics, Cardiology and Hematology, Jichi Medical School, Kawachi-gun, Tochigi 329-0498, Japan.
SOURCE: Journal of biological chemistry, (2001 Oct 19) 276 (42) 39012-20. Electronic Publication: 2001-08-06.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AB006972
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 20011029
Last Updated on STN: 20030105
Entered Medline: 20011204

AB The murine **sak** gene encodes a putative serine-threonine kinase which is homologous to the members of the Plk/Polo family. Although **Sak** protein is presumed to be involved in cell growth mechanism, efforts have failed to demonstrate its kinase activity. Little has been, therefore, elucidated how **Sak** is regulated and how **Sak** contributes to **cell proliferation**. Tec is a cytoplasmic protein-tyrosine kinase (PTK) which becomes activated by the stimulation of cytokine receptors, lymphocyte surface antigens, heterotrimeric G protein-linked receptors, and integrins. To clarify the in vivo function of Tec, we have tried to isolate the second messengers of Tec by using the yeast two-hybrid screening. One of such Tec-binding proteins turned out to be **Sak**. In human kidney 293 cells, **Sak** became tyrosine-phosphorylated by Tec, and the serine-threonine kinase activity of **Sak** was detected only under the presence of Tec, suggesting **Sak** to be an effector molecule of Tec. In addition, Tec activity efficiently protects **Sak** from the "PEST" sequence-dependent proteolysis. Internal deletion of the PEST sequences led to the stabilization of **Sak** proteins, and expression of these mutants acted suppressive to cell growth. Our data collectively supports a novel role of **Sak** acting in the PTK-mediated signaling pathway.

L20 ANSWER 4 OF 5 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
 ACCESSION NUMBER: 1999:453204 BIOSIS
 DOCUMENT NUMBER: PREV199900453204
 TITLE: Molecular analysis of selected cell cycle regulatory proteins during aerobic and hypoxic maintenance of human ovarian carcinoma cells.
 AUTHOR(S): Krtolica, A.; Krucher, N. A.; Ludlow, J. W. [Reprint author]
 CORPORATE SOURCE: Department of Biochemistry and Biophysics, University of Rochester School of Medicine and Dentistry, Rochester, NY, USA
 SOURCE: British Journal of Cancer, (Aug., 1999) Vol. 80, No. 12, pp. 1875-1883. print.
 CODEN: BJCAAI. ISSN: 0007-0920.
 DOCUMENT TYPE: Article
 LANGUAGE: English
 ENTRY DATE: Entered STN: 26 Oct 1999
 Last Updated on STN: 3 May 2000

AB We have previously reported on the development of an in vitro model system for studying the effect of hypoxia on ovarian carcinoma **cell proliferation** and invasion (Krtolica and Ludlow, 1996). These data indicate that the cell division cycle is reversibly arrested during the G1 phase. Here, we have continued this study to include the proliferation properties of both aerobic and hypoxic human ovarian carcinoma cells at the molecular level. The growth suppressor product of the retinoblastoma susceptibility gene, pRB, appears to be functional in these cells as determined by SV40 T-antigen binding studies. Additional G1-to-S cell cycle regulatory proteins, cyclins D and E, cyclin-dependent kinases (cdks) 4 and 2, and cdk inhibitors p27 and p18, also appear to be intact based on their apparent molecular weights and cell cycle stage-specific abundance. During hypoxia, there is a decrease in abundance of cyclins D and E, with an increase in p27 abundance. cdk4 activity towards pRB and cdk2 activity towards histone H1 are also decreased. Co-precipitation studies revealed an increased amount of p27 complexing with cyclin E-cdk2 during hypoxia than during aerobic cell growth. In addition, pRB-directed phosphatase activity was found to be greater in hypoxic than aerobic cells. Taken together, a model is suggested to explain hypoxia-induced cell cycle arrest in SKA human ovarian carcinoma cells.

L20 ANSWER 5 OF 5 MEDLINE on STN DUPLICATE 2
 ACCESSION NUMBER: 94294387 MEDLINE
 DOCUMENT NUMBER: PubMed ID: 8022793
 TITLE: **Sak**, a murine protein-serine/threonine kinase that is related to the Drosophila polo kinase and involved in **cell proliferation**.
 AUTHOR: Fode C; Motro B; Yousefi S; Heffernan M; Dennis J W
 CORPORATE SOURCE: Samuel Lunenfeld Research Institute, Mount Sinai Hospital, Toronto, ON Canada.
 SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1994 Jul 5) 91 (14) 6388-92.
 Journal code: 7505876. ISSN: 0027-8424.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 OTHER SOURCE: GENBANK-L29479; GENBANK-L29480
 ENTRY MONTH: 199408
 ENTRY DATE: Entered STN: 19940815
 Last Updated on STN: 20020420
 Entered Medline: 19940801

AB We have isolated murine cDNAs encoding two isoforms of a putative protein-serine/threonine kinase, designated **Sak-a** and **Sak-b**, which differ in their noncatalytic C-terminal ends. The kinase domain of **Sak** is related to the catalytic domains of the Drosophila polo, Saccharomyces cerevisiae CDC5, and murine Snk and Plk kinases, a family of proteins for which a role in controlling **cell**

proliferation has been established (polo, CDC5) or implicated (Snk, Plk). Northern and in situ RNA analyses of **Sak** gene expression in mouse embryos and adult tissues revealed that expression was associated with mitotic and meiotic cell division. In addition, during embryogenesis, **Sak** expression was prominent in the respiratory and olfactory mucosa. The pattern of **Sak** expression and its sequence homology with the polo gene family suggest that the **Sak** kinase may play a role in **cell proliferation**. In support of this, cell growth was suppressed by expression of a **Sak**-a-antisense fragment in CHO cells.

=>

CESSION NUMBER: 2002104290 EMBASE
TITLE: The Haemophilus ducreyi cytolethal distending toxin
activates sensors of DNA damage and repair complexes in
proliferating and non-proliferating cells.
AUTHOR: Li L.; Sharipo A.; Chaves-Olarte E.; Masucci M.G.; Levitsky
V.; Thelestam M.; Frisan T.
CORPORATE SOURCE: T. Frisan, Microbiology/Tumorbiology Center, Karolinska
Institutet, Stockholm, Sweden. teresa.frisan@mtc.ki.se
SOURCE: Cellular Microbiology, (2002) Vol. 4, No. 2, pp. 87-99.
Refs: 42
ISSN: 1462-5814 CODEN: CEMIF5
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20020328
Last Updated on STN: 20020328

AB Cytolethal distending toxins (CDTs) block proliferation of mammalian cells
by activating DNA damage-induced checkpoint responses. We demonstrate
that the Haemophilus ducreyi CDT (HdCDT) induces phosphorylation of the
histone H2AX as early as 1 h after intoxication and re-localization of the
DNA repair complex **Mre11** in **HeLa** cells with kinetics
similar to those observed upon ionizing radiation. Early phosphorylation
of H2AX was dependent on a functional Ataxia Telangiectasia mutated (ATM)
kinase. Microinjection of a His-tagged HdCdtB subunit, homologous to the
mammalian DNase I, was sufficient to induce re-localization of the
Mre11 complex 1 h post treatment. However, the enzymatic potency
was much lower than that exerted by bovine DNase I, which caused marked
chromatin changes at 10(6) times lower concentrations than HdCdtB. H2AX
phosphorylation and **Mre11** re-localization were induced also in
HdCDT-treated, non-proliferating dendritic cells (DCs) in a
differentiation dependent manner, and resulted in cell death. The data
highlight several novel aspects of CDTs biology. We demonstrate that the
toxin activates DNA damage-associated molecules in an ATM-dependent
manner, both in proliferating and non-proliferating cells, acting as other
DNA damaging agents. Induction of apoptotic death of immature DCs by
HdCDT may represent a previously unknown mechanism of immune evasion by
CDT-producing microbes.

=>

ACCESSION NUMBER: 2002083469 MEDLINE

DOCUMENT NUMBER: PubMed ID: 11809878

TITLE: Reconstitution of the mammalian DNA double-strand break end-joining reaction reveals a requirement for an **Mre11**/Rad50/NBS1-containing fraction.

AUTHOR: Huang Juren; Dynan William S

CORPORATE SOURCE: Institute of Molecular Medicine and Genetics, Program in Gene Regulation, CB-2803, Medical College of Georgia, 1120 15th Street, Augusta, GA 30912, USA.

SOURCE: Nucleic acids research, (2002 Feb 1) 30 (3) 667-74.
Journal code: 0411011. ISSN: 1362-4962.

PUB. COUNTRY: England: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200202

ENTRY DATE: Entered STN: 20020128

Last Updated on STN: 20030218

Entered Medline: 20020222

AB The non-homologous end-joining pathway promotes direct enzymatic rejoining of DNA double-strand breaks (DSBs) and is an important determinant of genome stability in eukaryotic cells. Although previous work has shown that this pathway requires Ku, DNA-PKcs and the DNA ligase IV/XRCC4 complex, we found that these proteins alone did not promote efficient joining of cohesive-ended DNA fragments in a cell-free assay. To identify factors that were missing from the reaction, we screened fractions from **HeLa** cell extracts for the ability to stimulate the joining of cohesive DNA ends in a complementation assay containing other known proteins required for DNA DSB repair. We identified a factor that restored end-joining activity to the level observed in crude nuclear extracts. Factor activity copurified with Rad50, **Mre11** and NBS1, three proteins that have previously been implicated in DSB repair by genetic and cytologic evidence. Factor activity was inhibited by anti-**Mre11** antibody. The reconstituted system remained fully dependent on DNL IV/XRCC4 and at least partially dependent on Ku, but the requirement for DNA-PKcs was progressively lost as other components were purified. Results support a model where DNA-PKcs acts early in the DSB repair pathway to regulate progression of the reaction, and where **Mre11**, Rad50 and NBS1 play a key role in aligning DNA ends in a synaptic complex immediately prior to ligation.

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	113824	hitoshi.in. or demo.in. or jenkins.in. or Rigel\$.As.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L2	105	mre11	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L3	2	L1 and L2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L4	45373	"cellular proliferation" or "cell proliferation"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L5	35	L2 and L4	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L6	456924	(system or method or process) WITH (identification or identifying or isolation or isolating)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L7	33	L5 and L6	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L8	105	"mre11"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L9	33	L7 and L8	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L10	3	NBS1 WITH RAD	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L11	1	L2 SAME (NBS1 and RAD)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L12	2546	augustus.in. or endress.in. or soppet.in. or hooykaas.in. or attikum.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09

L13	1	L12 and L2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L14	20329	engelhard.in. or morris.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L15	0	L14 and L2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L16	0	L14 and L8	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L17	26	L14 and "carcinoma associated"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L18	80335	"drug screen" or "therapeutic agent"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L19	32	L2 and L18	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L20	50	L12 and "anti-neoplastic agent"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L21	6	L20 and "signature set"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L22	0	youn.in. and augustus.in. and weaver.in. and endress.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L23	2	young.in. and augustus.in. and weaver.in. and endress.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L24	9	"WO 01/94629"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09

L25	1	"WO 200194629"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L26	4	loring.in. and kaser.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L27	36716	PC3 or HI299 or MDA or MB-231 or MCF7 or A549 or hela	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L28	41	L27 and L2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L29	113824	hitoshi.in. or demo.in. or jenkins. in. or Rigel\$.As.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L30	105	mre11	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L31	45373	"cellular proliferation" or "cell proliferation"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L32	456924	(system or method or process) WITH (identification or identifying or isolation or isolating)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L33	105	"mre11"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L34	2546	augustus.in. or endress.in. or soppet.in. or hooykaas.in. or attikum.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L35	20329	engelhard.in. or morris.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L36	0	L35 and L30	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09

L37	0	L35 and L33	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L38	80335	"drug screen" or "therapeutic agent"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L39	50	L34 and "anti-neoplastic agent"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L40	0	youn.in. and augustus.in. and weaver.in. and endress.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L41	36716	PC3 or HI299 or MDA or MB-231 or MCF7 or A549 or hela	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L42	1	L30 SAME (NBS1 and RAD)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L43	1	L34 and L30	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L44	20311	morris.in. or engelhard.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/04/15 15:09
L45	1	"WO 200194629"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
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L49	33	L48 and L33	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
L50	3	NBS1 WITH RAD	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/04/15 15:09
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L62	149	l59 and "multidrug resistance"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/04/15 15:25
L63	1	l62 and mre11	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/04/15 15:25
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L65	1	l62 and xrs2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/04/15 15:26
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L67	18	"hooykaas.in" or bundock.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/04/15 15:31
L68	1	l67 and mre11	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/04/15 15:27
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L71	0	l69 and "mdd"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/04/15 15:32
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L73	0	jacksong.in. and "MDDT"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/04/15 15:39
L74	16	jackson.in. and "MDDT"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/04/15 15:40
L75	16	l74 and (screen (s) compound)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/04/15 15:41
L76	1	l74 and (screen with compound)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/04/15 15:56
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... (1996) Human Rad50 is physically associated with human Mre11: Identification of ... anticancer **drug screen**. Proc Am Assoc Cancer Res 41:471. ...

cancer.ucsd.edu/howelllab/YeastMutants.pdf - [Similar pages](#)

Yeast Mutants As a Model System for Identification of Determinants ...

... The product of rad32Sp/MRE11Sc has nuclease and double-strand DNA ... A new world wide web site provides data from the NCI yeast anticancer **drug screen**. ...

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TUMOR CELL DEATH INDUCED BY TOPOISOMERASE-TARGETING DRUGS - Annual ...

... 134) have suggested that the Rad50/Mre11/Xrs2 complex may be involved in ...

Anticancer **Drug Screen**: multifactorial relationships with topoisomerase I, ...

arjournals.annualreviews.org/ doi/full/10.1146/annurev.pharmtox.41.1.53 - [Similar pages](#)

Current Opinion in Oncology - Fulltext: Volume 17(1) January 2005 ...

... et al: Mutations of an intronic repeat induce impaired MRE11 expression in

primary human ... Holbeck SL: Update on NCI in vitro **drug screen** utilities. ...

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... Adenovirus oncoproteins inactivate the Mre11- ... Neurologists strike gold in **drug screen** effort, News, Abbott, A., 417, 109 ...

www.nature.com/nature/archive/indexes/nature_415-420_subject.pdf?file=/nature/journal/v426/n6968/index.html

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... Protein Levels And Chemo-Sensitivity Patterns In Cell Lines Of The NCI Anticancer

Drug Screen. ... the repair of the DNA-dsbs (ie, DNA-PK, and RAD50/MRE11 proteins ...

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Cancer Research -- Jacob et al. 61 (17): 6555

... is part of the MRE11/RAD50 complex known to participate in DSB repair. ...


of the National Cancer Institute anticancer **drug screen** and correlations with ...

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... MCF7, another breast carcinoma from the NCI Anticancer **Drug Screen**, undergoes cell ... Phosphorylation of Histone H2AX and Activation of Mre11, Rad50, ...

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... amount suitable to complement and enhance the activity of the test compound. ... TRF2 associates with the MRE11-RAD50-EBNA1 facilitated recombinant double strand ...

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... RAD50/MRE11/microhomology-mediated end-joining or homologous recombination.

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The ATP switch model for ABC transporters - Nature Structural ...

... Elegant studies of the mammalian **multidrug resistance** P-glycoprotein (P-gp)

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... Ford and Higgins (1997) Structure of the **multidrug resistance** P-glycoprotein

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... For example among the RAD52 epistasis group (MRE11-RAD50-XRS2, RAD51, 52, ...

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... Examining the Role of Msh2 and **Mre11** in Telomere ...

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... and SN-38 by the **multidrug resistance** protein (MRP) and its inhibition by

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Sin, and tomosyn and decreased cathepsin L, **Mre11**, and topoisomerase II alpha. ...

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





... **measure** exponential growth rates directly (see part A of the ... function in **multidrug resistance**, our data suggest such ...

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... In a transgenic **functional assay**, the proline-rich C-terminal domain is not

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1: Biochim Biophys Acta 2001 Dec 30;1522(3):175-86 The modulation ...

... defective cell-cycle delays after induction of DSBs in the absence of **Mre11**.

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... damage that are mediated by the RAD50-MRE11-p95 complex. ... constituted the basis for

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... group by a **functional assay** or by the detection of a specific mutant allele,

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